Recovery Kernel

DISCONTINUANCE PROTOCOL: METHAMPHETAMINE

The Crash, The Timeline, and The Way Come Back To A Working Dopamine System

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Dis guide follow same structure with Recovery Kernel's Forced Discontinuance series — safety framework, genetic cross-reference, use-pattern classification, recovery protocol, and full protocol map — adapt am to wetin methamphetamine discontinuation really be. E no be seizure risk like way benzodiazepine withdrawal be. E na, for Recovery Kernel own words, 'one of the most profoundly unpleasant biological experiences' person fit go through, and e carry real and serious risk of its own: di crash produce depth of depression and anhedonia strong enough to be genuine suicide risk window. Dis guide dey treat dat risk with same serious way say e give seizure for benzo guide.

Section 0 — Safety Wey You Must Read First

⚠ KAIUTION

METHAMPHETAMINE WITHDRAWAL NO GO KILL YOU THROUGH SEIZURE LIKE BENZODIAZEPINE OR ALCOHOL WITHDRAWAL FIT DO. BUT THE CRASH WEY FOLLOW AFTER HEAVY OR LONG TIME USE E GIV ONE OF THE MOST BAD PSYCHOLOGICAL STATE FOR ADDICTION MEDICINE, AND THE DEPRESSION E GIVE NA REAL SUICIDE RISK WINDOW. TAKE THE CRASH AS SERIOUS LIKE ANY PHYSICAL WITHDRAWAL WEY TALK ABOUT OTHER PLACE FOR DIS LIBRARY.

Call emergency help sharp-sharp if you see:

- If you dey think say you wan die or do yourself harm, or if you feel say your depression pass wetin you fit handle — call or text 08062106493, or text HOME to 741741, both available 24/7.
- Chest pain, heart beat dey shake, breath no sweet, head pain wey come sudden pass, or face don drop/arm weak — dis fit show say you get problem for heart (cardiac event) or stroke, both na real danger after use heavy stimulant, and make you call 112 sharp sharp.
- Paranoid delusion, hallucination, or no sabi if wetin true—meth-induced psychosis fit remain for day to week after last time you use, e need doctor check.
- Wahala pass, or feeling say you no fit control wetin you dey do

IBELE

The crash itself — sleep plenty, sleep too much, hunger pass level, and wave of depression — e be thing wey dey come, e no be medical emergency by itself. E mean body start to recover. The difference wey matter na same one wey SSRI guide talk: mood and safety, no be discomfort. If you doubt, reach out.

Section 1 — Wetin Make Dis Guide Dey

The Recovery Kernel's own meth guide talk say dis one clear: 'nothing don go wrong. Your brain dey reset. The pain na signal say repair don start.' Dat sentence be the main idea behind this whole discontinuance protocol. Meth withdrawal no get chemical antidote and no safe substitute-and-taper strategy like way benzodiazepines or opioids do — no be anything like cross-taper here. Wetin we get instead na well-documented, predictable biological timeline, and knowing dat timeline before time come na one form of harm reduction: e be difference between 'dis pain mean say I need more meth to feel normal' na 'dis misery na Day 3 of process wey dem record say e go end.'

Short Story: Wetin Happen When Dem Discover Danger

Methamphetamine na first time dem synthesize am from ephedrine for 1893 by Japanese chemist Nagai Nagayoshi, and later crystallize am into form wey you fit recognize till today for 1919 by Akira Ogata. Dem patent am and sell am for Japan for the 1920s under brand name Philopon, dem market am for fatigue, narcolepsy, and weight loss — na proper pharmaceutical product, e no sabi say e dangerous yet.

E change when dem scatter am for World War II. German chemist dem wey name Temmler patent their own methamphetame formulation for 1937, and by 1938 e dey sell pass counter under brand name Pervitin — distribute to Wehrmacht soldiers plenty plenty (estimate talk say tens of millions of tablets) specifically to keep the sleepless, forced-march pace of the Blitzkrieg, and to civilian factory workers to sustain long shifts. Japan run like program, give methamphetame to kamikaze pilots and to civilian factory workers for same reason: e let tired people continue work pass the point dem body no go let dem pass am.

THE CENTURION

Dis na history background of di exact pattern for Section 5 of dis guide — use stimulant make person carry out work pace wey e no fit sustain. E be true for tired soldier and factory worker for 1940, and e still be true for same pattern today.

The danger don become clear for time wey follow. Japan especially pass through big epidemic postwar when dem scatter military stockpile go civilian market — by some estimate, small percentage of people dey misuse the drug inside few year after war end. The size of addiction, psychosis, and body break down wey e do was

too much say Japan pass Stimulant Control Law for 1951, one of first drug-specific stimulant rule for world — direct law response to wetin no control access don show at national level. Wetin take global war come... na na epidemic wey dem use establish for 1940s, e like small version of wetin dis guide Section 2 timeline dey try teach make we know before: power of this drug go scatter direct with how much body dey ask to run on am without stop.

Section 2 — Di Time Wey Body Go Start Waka Off

Time chart	Kwetin be wetin dey happen biologically?	Kedu be feeling?	Wetin I do?
Hours 0–24 (The Crash)	Dopamine reserve don finish; receptor signal dey fall	Waka-waka die, sleep pass, stomach dey cry, low mood, worry plenty	Sleep. Chop food. Drink water. No fight the crash — na body dey start recover. Electrolytes.
Day 1–5 (Na Pit)	Dopamine don fall ground; D2 receptors still down low; e system dey, like say e dey shout for di chemical wey e adapt round.	Heavy depression, no fit enjoy anything (no sabi joy), tire pass, craving strong, sleep scatter, head dey foggy	Dis na highest time wey you fit relapse and high risk of suicide for dis whole guide. No dey alone if e fit avoid. Text 08062106493 if crisis. Start di Tier 1 supplement support (Section 8).
Day 5–14 (The Gray)	Symptoms don start better; dopamine system still weak pass; receptor recovery just begin small small	Constant flat feeling, low drive, sleep no dey settle, brain weak, craving come and go	Start small exercise — dis na repair signal (Section 3). Begin identify and avoid environment use-triggers.
Week 2–4 (Di Slow Come Back)	DRD2 receptor density start dey show small sign of recover; dopamine synthesis slow-slow dey rise; prefrontal cortex repair don begin	Mood dey improve slow-slow, no straight line; brain fog don lift; craving no be constant thing again, e dey come and go; sleep don settle down.	Set steady daily pattern. First proper goal wey you wan achieve go come real here, no be before.
Month 1–3	Measure d2 receptor recoveri; rebuild grey matter for prefrontal cortex; make dopamine synthesis normal	Real improvement with craving wey dey come sometimes and mood change pass; PAWS episode fit happen	Identity reconstruction work make sense here — no before. Na when power really start come back.
Months 3–12	Big receptor recover; BDNF-drive	Mood don reach near baseline passu funfun;	Keep every protocol wey dey follow. Guard

Time chart	Kwetin be wetin dey happen biologically?	Kedu be feeling?	Wetin I do?
	neuroplasticity dey rebuild damaged dopamine axon well-well; prefrontal cortex function don return plenty	craving na say e come from context/cue, no be say e just happen; brain work don return well-well; real joy for life go become easy to reach.	dis window from big stress event if e fit — di system don build back, no reach full structure strong pass yet.

Section 3 — Wetin Go Take Time Till Brain Don Return To Normal

Dis na question wey dey underneath every stage of timeline above, e deserve direct answer pass vague reassurance. True answer get two part: crisis go settle for few weeks, but full receptor-level recovery take plenty time — and knowing the real number change wetin 'normal' suppose be like at each stage.

THE CENTURION

Di specific data point, wey dem talk direct from neuroimaging research wey Recovery Kernel self dey use (Volkow et al.): dopamine D2 receptor recovery after methamphetamine stop na significant, but substantial recovery take 12 to 17 months of sustained abstinence. E slow pass wetin I go tell you say e measurably slower than alcohol (significant D2 receptor improvement by month 1, per Heinz et al.) or cocaine — methamphetamine dey cause the most extensive dopamine terminal damage among common stimulants, and di repair timeline show dat.

Within dat bigger 12-17 month time frame, there get one faster, more sharp rebuilding window wey you need know about specially: the first 90 days. The Recovery Kernel's neuroplasticity framework show dis well-well — days 31 to 60 na when new neural pathways start form and old ones begin weak; days 61 to 90 na when those new patterns start solidify into change wey you fit measure structurally. By 90 days, with consistent exercise, sleep, and the other neuroplasticity things for Section 8 below, dopamine receptor density don improve pass, gray matter volume don increase wey you fit see, and cravings don reduce plenty for most people — even say di full recovery wey dey 12-17 months still dey happen under dat.

Milestone ni Recovery	Timeline wey e real
Acute crash resolve	Day 1–5
Cravings change from dey always there to dey come when something trigger am	Week 2–4
First measurable D2 receptor recovery for imaging	Week 2 – Week 4 pass Month 1
90 days neuroplasticity window don close — new habit structure don cement well well	Day 90

Milestone ni Recovery	Timeline wey e real
Mood and thinking wey dey normal pass for most people	Months 3–12
Substantial D2 receptor recovery (di headline number)	12–17 months

Kedu be hearing

If you dey month 4 and still dey have dull, gray day, e no mean say process don fail or say you broken for way wey timeline no fit see am. According to data above, you dey inside genuine recovering system — 'near-baseline' at months 3-12 mean exactly dat: closer, no mean finished. Finish line for deepest layer of this repair na closer to year and half pass season. Dat no be number wey go scatter spirit. E be honest one, and e mean say dull day you still dey have na expected, no be sign say you fail.

One Honest Warning: Dis Timeline Dey Assume Say You No Drink At All

Every number for dis section — di weeks, di months, di 12-17 month headline figure — na people wey stop methamphetamine and stop there. For real life, plenty recovery no look like dat clean. Two substitution common enough to name specifically, because each one interact with the exact repair mechanism dis section talk about, and each one fit slow down time rather than be neutral bridge through am.

Swap	Wetin e really do give recovery mechanism?	Likely effect for timeline
Caffeine (wey dem use manage tiredness and no-want-do phase for Section 2)	Caffeine no dey drain dopamine like methamphetamine do, so e no be 'the same thing in miniature.' But e go stimulate the HPA axis — add cortisol-system activation on top of a system, per Section 6, wey fit already dey run hot (especially for di Met/Met 'Worrier' profile) — and heavy or late-day caffeine use directly spoil sleep quality and how long you sleep. Section 8's Tier 3 clear say sleep na time say BDNF synthesis and dopamine-system repair reach peak.	E go stretch time for you indirect way, by spoil di sleep quality wey proper repair work depend on, e no be say na direct dopamine mechanism. E more caffeine wey dem substitute specifically to make person pass through di fatigue Section 2 describe, e mean say e dey fight di exact process wey fatigue part of.
Alcohol (use pass sleep)	Dis na more troubling substitution of the two. Alcohol dey make you sleepy, but e scatter sleep structure and specifically suppress REM sleep — and REM na, according to dis library own material, exactly di sleep stage wey 'emotional memory processing and dopamine system maintenance happen.' Use alcohol to force sleep start while suppressing	E fit prolong timeline direct, through REM sleep wey don spoil, and e introduce second risk say dis section numbers no account for at all: new dependence dey form on top of one wey dey recover — genuinely likely for DRD2 A1

Swap	Wetin e really do give recovery mechanism?	Likely effect for timeline
	di sleep stage wey most depend on recovery na close to solve di visible problem (no fit sleep) while worsen di invisible one (di sleep wey supposed to repair di dopamine system no dey actually happen).	allele / Reward Deficiency Syndrome profile for Section 6, where reward circuit already ready to over-attach to whatever substance dey reliably available.

⚠ KAIUTION

Neither of these substitution no dey show here as moral failure — reach for caffeine or drink to pass through unbearable time na very understandable response to real suffering. But the true version of this guide must say plain: neither one be neutral bridge through timeline for table above. If sleep be problem, di non-alcohol tools for Section 8's Tier 3 (melatonin, magnesium glycinate, glycine, consistent schedule) dey work with repair mechanism instead of against am.

Na wa o: Why "One Year Break, Then I Go Better" No Too True

There be idea wey need talk about straight, because e sound like correct conclusion wey come from number for Section 3 tin self, and e no too wrong altogether — which na wetin make am dangerous. Idea na: 'if D2 receptors recover well-well by 12-17 months, I fit take year off, let my brain heal, and after dat know how to use safe again, or start from zero.' Old way wey dem dey talk about addiction treatment — 'the moment you pick up again, you instantly back to full addiction' — e no be 100% true. But the feeling say long break go reset you to zero, e no be 100% true. Either. Truth tin dey for middle, and break down na really good way to see wetin happen.

Kedu be true (like bone wey don heal)?	Wetin also true (Why gait still change)
Bone no dey heal. D2 receptor density, according to data for Section 3, really go recover for proper, measured time—dis no be sweet talk or just hope story, na wetin imaging show.	Bone wey don heal no be like bone wey no break. E go reshape round di place wey e get wahala. E fit carry real weight again — but e no automatically give you same load, at same angle, same way, if body no learn again how.
Tolerance na thing wey e truly lose when you no use anything at all — dis na pharmacology wey dem write am clear, no be guess work.	Lost tolerance i mean system wey before sabi take dose one kind, now e no fit take dose same way safely. If you return to amount wey you know, after you don break for real, na like giving body wey capacity change, even though memory say wetin normal be, e still dey there. E be precisely mechanism behind high overdose and heart-event risk (Section 0) wey people see especially when dem relapse after long time no use — e no because drug don strong pass, but because body's power to take

Kedu be true (like bone wey don heal)?	Wetin also true (Why gait still change)
	am don small while expectation no change.
Di old way wey dem dey frame say e be 'instant relapse to full addiction' e too much for say neurology na wetin fix well-well and never go change.	E no be say e baseless o. Section 6 material wey talk about epigenetics dey show how gene expression for reward-circuitry change with repeated use — and those shifted pathways no mean say dem need to build from zero to restart. Path wey don carve before, person fit walk am again faster pass say na new path dem go carve first time. Na this kind reason say relapse after break often blow up pass how e start, even when receptor measures return to normal level.

⚠ KAIUTION

Make e clear: period of real abstinence no be reset button, and e no be useless too — both of the ways we talk about for introduction to this section na partly correct and partly wrong at same time. Wetin e actually do na system wey don repair like way Section 3 dey describe, while still different structure from one wey dey before use start — different tolerance, different vulnerability to fast reactivation, different cardiovascular capacity. Try to put same weight on am, same way, na where danger gather. Dis no be technical note. E be say relapse after proper break carry own different kind, high-risk profile pass say e just 'safe now' or 'straight back to zero'.

One popular lie wey need correct: 'Your Cells Go Change Whole Thing Every 7 Year'

Dis idea dey circulate everywhere, including inside recovery communities specially — but e no stand with wetin dem actually measure, and di corrected version better pass di myth, no less. Different tissues change over at very different speed: skin and gut lining for days to weeks, fat cells for roughly 10% every year, skeleton for roughly one decade. But di tissue wey matter most for everything inside dis guide — neurons for cerebral cortex — dem no get meaningful replacement at all after early development. Di neurons wey dey involved for reward pathways say this whole guide describe na, For most part, na same body cell wey you get before you ever use meth. Dem no dey swap am for new one anywhere time, whether seven year or no.

Wetin age and time give? Two real thing, different from myth. First, wetin dey recover for timeline for dis guide na no be new cell dey replace old one — e be density, sensitivity, and expression of receptors on the neuron wey already there, plus di synaptic connection between dem (material epigenetics for Section 6). Second, aging e add real complication layer, through different and well-document mechanism: cellular senescence. As body dey age, some cell — including supporting glial cell for brain — stop dey divide but remain metabolically active, dey secrete inflammatory signaling molecule wey dey gather over time and

contribute to neuroinflammation for background. Dis na real thing, current addiction and aging science, and e mean say recovering system no be fixed target — e dey age for background while e dey repair, which fit make whole process feel harder or slow pass the more time and the more use don pass, independent of dopamine-specific timeline for Section 3.

Kedu be say you dey hear?

Na so kind your point wey survive correction, and e fit be pass original: because same neurons dey remain instead of change, di old pathways dis guide talk about — di fast reactivation risk, di learned cue responses — dem no disappear after one year off. Dem still physically there, for same cells, just quiet small. Na that exactly why write off one year of real work say 'e no work' or 'I no strong enough' or reach for 'once addict, always addict' na wrong conclusion to draw from relapse. — but e be like say person dey assume say year go wipe pathways wey no even dey plan to delete from start, just make dem pass competition. The real work never be full erase. E be build new, competing pathways strong enough say the old ones stop being the easiest way — which na exactly why Section 11's Protocol 20/C-28 material talk about redirecting this exact reward circuitry toward genuine, high-intensity natural alternatives, instead of just ask the circuitry itself to vanish. And e good to tell make we clear: go back to substance after time away no always mean say person decide say e fit... Na true thing say person just curious — dey wonder, dey test, just dey like wetin one thing—na real and e fit understand for itself, no need make you tie am with story about will power or say you fail morally.

Section 4 — Wetin You Go See Pass

Timeline for Section 2 e tell you wetin dey happen biologically and roughly when. Dis section different — na attempt to actually describe wetin e feel like from inside, pass deep pass wetin table row fit hold, so say when e happen, you go recognize am instead of panic over am.

Anhedonia — na e don flatten pass say e be hardest symptom

Anhedonia dem talk am like clinical pass for most material wey dey talk about withdrawal — 'inability to feel pleasure' — like say na dat one be all say e be. E no be. Wetin e actually feel like, for most people, na like watching your own life through glass: food no taste like anything, music you use love before register as sound without the feeling wey dey attach, joke land as information rather than something funny, person wey you love no give the warmth say being near dem used to give. E no mean say you stop caring. Na say the machine wey use translate 'good thing happening' into 'felt Good' dey run for small small pass normal signal, because dat machinery na the exact dopamine system say methamphetamine hijack and empty am. Dis genuinely one of di most disorienting part of whole process, because e fit bring second-order fear on top of di flatness itself — 'wetin if I never feel nothing again' — and dat fear na thing wey must name straight:

according to Section 3 data, dis na documented, temporary state with known recovery arc, no be permanent personality change.

Other symptom wey sabi name clear clear

- Hypervigilance and feeling like say something dey run pass, restless thing even when you don tire pass — your nervous system no fit slow down like body need sleep.
- Nwafo, craving wey strong die, wey come without warnin', e fit start from wetin no sweet at all — smell, song small, text message alert — and say for moment na only thing wey go make you pass this time.
- Wahala small small, na kind hot temper wey fit shock you and people for your surround, especially when you dey for Pit phase (Day 1-5).
- Real cognitive fog — no fit find word, struggle follow talk, feeling like you dey think for inside mud. Dis no be sign say na permanent wahala; e follow same recovery path with everything else for Section 2 and 3.
- Vivid, sometimes disturb dream when sleep start come back — na brain dey do plenty catch-up REM processing after time wey sleep don scatter or no come well-well.

Dis one sabi take eye well-well, because e often be di symptom wey most confuse pass di one wey clearly dey cause wahala. Di same synaptic strengthening wey BDNF dey drive wey dey repair dopamine system (Section 3) e work partly through glutamate signaling — glutamate and NMDA receptor activity na normal, necessary part of how synapses actually get stronger, no be pure toxic byproduct. So di intense dream activity for dis recovery window e best understand as dual causation: real repair work dey happen, with unusually vivid and frequent dreaming as side effect of dat work, no be primary sign say damage still dey go on. For Some people e go pass just vivid — dey dream strong enough to wake up from am physically, sometimes plenty time through night, which turn come own sleep-disruption problem wey dey build on top of insomnia wey already dey. Others notice say dem suddenly remember dreams with detail dem normally no go — wake up and fit talk about dream long long, sometimes catch self talk about am like say e be thing wey really happen, before the difference settle back down. Both of these na consistent with REM rebound and synaptic remodeling wey dey happen during this time, no be sign say something E don run wrong with your hold for real life.

Kedu be say you dey hear?

If you read di anhedonia description above and think 'na so e be, and I no know say word fi talk am' — dat reaction na common, and e good make you pause on am. Plenty people go pass dis particular symptom feeling like something don wrong with dem alone, especially because nobody describe am well-well enough for dem see say na one wey everybody wey dey pass dis go experience. E get name. E get timeline. E no be you.

Wetin Make Dis One Hard Pass: Di Things Wey You Must Avoid On Purpose

Some wetin go happen during dis recovery na go happen no matter wetin happen — di timeline for Section 2 and Section 3 e be say e fixed biology pass. But some wetin make person feel like e wan die, make e drag am back to use, e no be di underlying repair process tin itself — na specific, things wey fit avoid, wey dem stack on top of am wey e make window wey already hard, become harder pass. Knowing which one na which matter, because e easy too confuse 'dis don worse cause of wetin I eat/drink/no sleep' for 'dis recovery no dey work,' when actual answer na often much more fit fix.

Wetin make e worse?	Why?
Food wey sugar high and glycemic high	Blood sugar spike and crash dey make mood unstable pass, e go add to energy crash wey dey happen already from dopamine depletion — na crash for top of crash.
Kaffeine na wetin alcohol dey do?	E don cover well well for Section 3 up there — caffeine indirect way, through make you no sleep well and make your HPA-axis scatter; alcohol direct way, through e suppress REM on top of the REM rebound wey dey happen already.
Nicotine and other stimulants	Di clear one, but e good make I talk am plain: any stimulant, including nicotine, dey keep same reward and arousal circuitry busy wey dis whole recovery na try to let settle down.
Bad sleep habit — screen till late night, time wey you dey sleep no steady	E dey directly undermine di way body repair wey need sleep (BDNF make, memory strong up) wey Section 8, Tier 3 show say na main thing wey dey drive how fast you go recover for Section 3.
Dehydration na wetin body no get water enough, e be like say you no drink enough water. Skipped meals na when you no chop food for time wey you suppose chop.	Brain wey dey do dis kind repair work need steady glucose and water; run on empty go add another layer of drain for top of wetin body dey pass already.
Long time no see / Long time alone	E no be food or thing wey you go chop, but e good make we talk about am — isolation na wetin go make every withdrawal syndrome wey dis library record pass, e worsen pass, and e remove support contact wey Section 0 know say e dey protect during time wey e high-risk pass.

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Exercise na clear exception zu list dis, e deserve make we name am one rather than just throw am for caution about overdo things. For Section 8's Tier 3, e be primary way we dey generate BDNF wey dey drive the whole recovery timeline for Section 3 — more of am, no less, na general direction we need to go as capacity allow.

Kedu be say you dey hear?

E choke say almost everything wey dey pain for this recovery — na flatness, na fatigue, na intense dreams, na cravings — na your body dey actively repair itself. E no be sign say wetin don wrong. E easy die, for middle of am, make you confuse the discomfort of repairing with evidence say recovery no dey work, and make you reach for drug just to make that idiopathic-feeling wave of symptoms stop — e no be say craving itself don strong pass, but because nobody tell you say wetin you dey feel na expected, temporary, and productive. Knowing dat one before no Make dis discomfort vanish. But e change wetin be dis discomfort mean, and dat change wetin you go do about am.

Section 5 — Di Way We Live For Work And How Dreams Meet Waking Life

E no be say everybody wey develop methamphetamine dependence start from party or crisis. One real and common pattern na use wey start, or grow pass, specifically for work — long shift, multiple job, caregiving on top of full workday, kind of chronic overextension wey stop no feel like option. Stimulant effect of Methamphetamine fit look, for while, like only way to keep meet schedule wey never sustainable from beginning. Dis pattern deserve own mention because e produce specific and distinct symptom wey other description of meth use often miss entirely.

⚠ KAIUTION

Chronic, severe lack of sleep — kind wey come from use stimulant make person push pass sleep body supposed to get — fit cause one thing wey dream matter start enter waking mind: small, clear, like dream picture or feeling while you dey wake, feel say e no real, or moment wey you no fit know if wetin happen or na dream. Dis na well-known consequence of severe lack of sleep itself (sometimes dem call am hypnagogic intrusion or microsleep with dream content), e no be sign of psychosis by itself — though e share some feature with The psychosis risk wey dey talk for Section 0, and any version of dis wey include paranoid delusion or break from reality testing (instead of small, like dream-like feeling inside your head) must be check like say Section 0 describe am.

If dis pattern dey apply to you, recovery task include something wey rest of this guide no fully cover: actually fix the root cause of overwork, no just di substance. Build up dopamine system (Section 3) while you return to same unsustainable schedule wey help create di dependence for first place na build back into same condition wey break am. Dis one worth talk about honestly rather than leave e dey imply.

Section 6 — Gene Cross-Reference

Gene	Wetin e govern	Relevance here
COMT	Speed wey body clear	Val/Val ('Warrior,' fast clearance) na

Gene	Wetin e govern	Relevance here
(Val158Met)	dopamine	primary vulnerability profile ni for methamphetamine specially — e be di fast-clearin', intensity-seekin' system wey dey inside meth's extreme dopamine signal di first experience wey ever fully satisfy am. Recovery need genuine high-intensity alternatives, no just abstinence.
DRD2 / ANKK1 (Taq1A, A1 allele)	DRD2 receptor density	The A1 allele carrier's reward circuit dey run with low baseline D2 density — Reward Deficiency Syndrome. Dis na single most important genetic target for recovery supplementation (the 'Brain Stack' for Section 8), because e be the receptor system itself wey need to rebuild density, no just clear di drug.
MAOA (uVNTR)	How fast dopamine, serotonin, and norepinephrine dey break down?	Allele wey no dey do much, e go make monoamine gather; allele wey dey do much, e go scatter dem fast. Second pattern na e be say e relate to high impulse and how bad person fit use am — e matter for when you go take supplement and knowing say you fit relapse.
BDNF (Val66Met)	Neural repair and plasticity signaling	Met carriers dey secrete small BDNF when dem do activity. Increase for BDNF wey come from exercise na main tool for neuroplasticity for whole guide dis one—e work no matter genotype, but Met carriers fit need higher level of exercise to make same kind signal wey go repair. Dis one no be option for this recovery—na the repair system itself.
CYP2D6 / CYP2B6	Metabolism of some medication wey dey help with Recovery Kernel (like bupropion)	If dem introduce any medicine while e dey recover — poor metabolizer go risk poison for normal dose; ultra-rapid metabolizer fit no see nothing. E good make you tell doctor before you start anything.

How COMT Status Really Change How Crash Feel

Na COMT row di top dey talk say na genotype; dis one concern wetin e really look like every day, cause Val/Val and Met/Met dem go withdraw different way plenty say e worth describe am separate pass say make e remain just abstract name.

COMT Profile	Baseline Before Meth	How e crash show?
Val/Val ('Warrior') — clear dopamine	Sometimes when e no dey do anything, e go feel dull;	Crash na way e fit show like deep flat feeling and bore pass anxiety—like

COMT Profile	Baseline Before Meth	How e crash show?
quick-quick, make baseline dopamine low down	e go like things wey dey hot, new thing, and situation wey get big risk, even before e use any drug.	kind of grey, no stimulation empty feeling. Loss of motivation na time be biggest complain. Craving usually dey focus on want to feel something strong again, no be say escape trouble only.
Met/Met ('Worrier' / 'Sage') — slow dopamine clear, high baseline dopamine and stress-chemical vibe	E choke you with anxiety or overthink plenty when you resting, even before you take anything wey dey your body; your nervous system go try run 'hot'.	Crash na be say e go show with plenty anxiety, rumination, and agitation wey dey build on top of di flatness — no just low mood but one kind wired-and-tired feeling. Dis profile also more likely to get the hypervigilance wey talk for Section 4, and e fit catch di sleep-deprivation effect for Section 5 harder because stress-chemical level already high.
Heterozygous (Val/Met)	One level wey dey middle of the two profile diji.	Sometimes e mix of two pattern — some flat, some anxious — no be say dem too high or too low like di homozygous profile.

GABRA2 — 'Brakes' System and Why Stop No Be Same Thing For Everybody

Methamphetamine tin be drug wey no be GABA-A drug like benzodiazepines — but GABRA2 status still matter here, for different reason. GABRA2 control the density and sensitivity of brain's main inhibitory ('braking') system — general capacity to press brake on impulse, urge, and behavior once e don start already. Dis direct relate to how hard 'just stop' really be for person, no matter willpower.

GABRA2 Profile	Kwetin be say wetin e mean to stop?
Low-risk / normal GABRA2 function	Plenty more brake power wey dey stop you from do wetin your hand wan do — e shock wey come, e easy to cut am before e turn to action.
High-risk GABRA2 variant (thin baseline inhibitory tone)	Less braking capacity available before use ever starts — meaning gap wey crave and action dey genuinely short pass, e no easy to widen am with willpower alone. Dis na hardware difference, e no be character difference, and e be same GABRA2 architecture wey document for Recovery Kernel's GABA Braking Systems material for benzodiazepines and alcohol — e apply here too, just to impulse control generally rather than to a GABA-A drug specifically.

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If stopping don feel harder pass say e dey for you pass say person wey you know wey use same drug, same amount — dat difference fit be real hardware, no be gap for effort or commitment. GABRA2 status na exactly kind thing wey make 'just stop' be wildly different task for two different nervous system wey dey face same craving.

Pass The Gene Wey You Born With: E Meth Use Change Gene Expression?

Everything for table above concern genetic hardware person come world with. One different question wey dey grow better study na if methamphetamine use itself fit change how gene dey express — no be underlying DNA sequence, which use no go change, but the epigenetic marks wey determine say which gene go switch on or off, and how strong. Dis difference matter: nobody's genetic code no go rewrite by using substance, but the activity level of specific genes — especially dopamine-pathway and stress-response genes wey make we talk throughout this section — fit genuinely shift with sustained use.

Wetin don well-establish for addiction neuroscience generally (dis no be research wey talk about meth alone, but e include methamphetamine for literature wey e draw from): repeated exposure to stimulant dey cause build up of transcription factor wey call ΔFosB for reward circuitry inside brain. Dis one too change how many gene follow am wey dey involve with reward feeling and craving — dis na one of the most times dem don confirm for addiction molecular biology, e dey describe for foundational literature wey dis compendium's own bibliography cite (Nestler, 2001, on the molecular basis of long-term plasticity underlying addiction). Whether methamphetamine go create extra, drug-specific epigenetic signature pass wetin... Dis general mechanism — and if any of these changes fit pass down for generation after generation, like some animal study on other stress and substance don suggest — na area wey dey genuinely active research, e no fully settle yet. E go be overdo science to show dat second part as confirmed; na open question wey worth watch, e no be documented fact yet.

SANCTUARY

E be like say dis same epigenetic mechanism dey run both ways. Material wey Recovery Kernel give person about neuroplasticity from exercise dey talk 'the epigenetic feedback loop' direct — deliberate, matched physical activity go produce measurable changes for gene expression on a documented timeline (habit and identity shift by months 1-3, measurable epigenetic changes by months 3-6, new neurological baseline by month 6 and beyond). Whatever gene-expression change wey e produce, di Tier 3 recovery work for Section 8 no be just symptom management — na e dey work through the same biological channel, in opposite direction.

Who Dis Guide No Fit Make Safe: The Genetically Contraindicated Profile

Almost everything for this guide write say recovery, if e done well, e dey available to anybody no matter how your gene profile — the time fit hard or easy depending on your hardware, but e dey available. There one profile wey that assumption no dey hold, and na important make we talk am plain rather than leave am hidden inside genetics table.

🚨 BIG PROBLEM

For one specific genetic and psychiatric combination, meth use carry risk wey different kind, no be just level, from everything else for dis guide — include real risk say e fit trigger psychotic disorder wey no fully clear even if person stop using am. Dis dey explain well-well below.

Profile	Why dis one different pass kind, no be just degree.
COMT Met/Met ('Sage'/'Worrier' — slow dopamine clearance, high baseline dopamine and stress-chemical tone) combine with personal or family history of psychosis, schizophrenia, or bipolar disorder	Dis same logic wey Recovery Kernel's own material dey use e apply to most strong synthetic cathinones (MDPV, alpha-PVP): na pure dopamine-flooding stimulant, wey dem enter system wey already get high baseline dopamine tone and no get serotonin's tempering influence, e go give highest documented psychosis-precipitation risk for stimulant category. For most people, stimulant-induced psychosis — though serious — e go settle with abstinence over days to weeks. But for dis specific combination, the risk na different pass: make person wey get underlying vulnerability start first psychotic episode fit trigger condition wey no fully clear when drug don wash out, because the Drug fit be like trigger man wan start something wey dey inside, e no be only thing wey cause na temporary state.

How make we know dis before e too late?

Full genetic testing (COMT rs4680, DRD2/ANKK1 Taq1A) na most precise way to know, but e no dey available for everybody, and the more urgent and available screening tool na plain history — dis one worth check well-well honestly, ideally before any use, or as early as possible into recognizing a use pattern:

- Any time before, your mama, papa, brother, sister, or grandpa/grandma sabi say e get history for schizophrenia, bipolar disorder, or any kind of psycho episode?
- You ever personally sabi wetin happen to you, for any substance include drink or weed, wey e no just make you feel very different, but make you lose sense of wetin dey really true—no be just paranoia ma or anxiety, but truly unsure wetin dey happen around you?
- You dey feel like you wan scatter, tense, or too much energy from normal kind stimulation even without any drug — di Met/Met 'Worrier' pattern wey dem talk for Section 6 above?
- Anytime say psychiatrist or doctor raise talk about mood or psychotic-spectrum disorder, even one wey you no follow through or you disagree wid?

⚠ KAIUTION

Any combination of say 'yes' for family or personal history questions with the Met/Met stimulant-sensitivity pattern na one wey must treat as real contraindication, no be risk factor wey you go weigh against the appeal of the drug's effects. Dis na one place for

this whole guide wey message no be 'see how to reduce danger' — e be 'dis specific combination na why danger no fit fully reduce.' If dis describe you or person wey you dey try help, psychiatric evaluation before any further use — no after crisis — na responsible next step, and e be a E be very reasonable thing make you carry go see doctor direct: 'I wan sabi my personal psychosis risk kedu before I use dis again.'

Phenotyping Without Test Result

- Stimulants don give you feel like normal, focused, and alive for way anything else never do? → Wahabi Warrior COMT profile with DRD2 deficit for under.
- You use stimulant specially to do — work hard pass, long time, reach plenty? → Likely Val/Val COMT.
- You start with prescription stimulant, e follow up from there? → E likely be DRD2 deficit with tolerance develop.
- You sabi struggle with depression or emotional flatness even when no dey use? → Likely MTHFR involvement — methylation support (5-MTHF) na priority side by side with everything else for Section 8.

Section 7 — Use Pattern Classification

Pattern	Typical profile	How much pain you get?	Posture wey I recommend
Low	Sometimes time, no be binge use; drink inside mouth or sometimes puff up nose; use small small for week few.	Low — crash na real thing, but e usually short pass and no too bad	Cessation no bad if you do am by yourself, but get support contact still good make you feel better with this low mood. Safety sign for Section 0 still dey work.
Medium	Normal use, no be long time binge; week till few month use	Moderate — expect the full crash and pit phases for timeline for Section 2	Support structure abi na section 8 supplement stack from day one, e go reduce how bad thing dey.
High	Binge-crash cycling — period wey you dey use wetin many days follow by long time crash — for month	High — di binge-crash pattern wey e self dey accelerate dopamine system depletion pass wetin steady use fit produce	E strong say you need medical help when you dey pass through crash phase, especially for time wey e dey high risk of suicide inside Day 1 till Day 5. No do dis

Pattern	Typical profile	How much pain you get?	Posture wey I recommend
			one by yourself.
Chronic / IV or smoked way, long-term	Months to year wey you dey use, especially through smoking or injection (e faster, more deep dopamine spike with corresponding deep depletion)	Highest — dis na population wey dem measure di 12–17 month D2 Recovery Kernel timeline for for Section 3.	E strongy recommend make dem get full medical and psychiatric support when e happen sharp crash. E be also people wey go fit benefit pass form proper treatment engagement pass say na pure self-directing protocol.

Section 8 — The Recovery Protocol

⚠ KAIUTION

No be anything here dey treat suicidal thought, psychosis, or heart problem. If any Section 0 sign show up, dat one go take pass dis protocol.

Tier 1 — Biological Support

Two additions below talk straight to di glutamate side of this recovery, alongside NAC — meth's dopamine surge co-releases glutamate for cortico-striatal circuits, and excess NMDA receptor activation from dat glutamate na real contributor to di neurotoxicity for Section 3, and plausibly to di intensified, sometimes disturbing dreams wey show up as REM sleep rebounds once real sleep return (Section 4).

Support	Role
Tyrosine (500-1000mg, morning, far from protein)	Dopamine precursor — start when you dey chop regular food, usually Day 3-5.
NAC (600mg twice daily with food)	Dual-action: glutamate homeostasis and craving reduction via the cystine-glutamate exchange system, AND a glutathione precursor supporting liver detoxification — chronic meth use, especially combined with any adulterants in the supply, place real burden for hepatic detox pathways, and NAC replenish liver's primary antioxidant molecule.
NAD+ (nicotinamide adenine dinucleotide)	Chronic substance use na beji beji deplete NAD+, coenzyme wey dey center to cell energy make pass and DNA repair — e be part of why early recovery get such heavy tiredness. IV infusion na most effective and most study way to carry support for acute withdrawal specifically, with some clinical program talk say dem

Support	Role
	see good reduction for withdrawal pain; nasal spray, sublingual, and mouth precursor (NMN/NR) forms dey easy pass but no direct like that. Dis one be real option, e no be first-line must-have — the basic work below (sleep, exercise, nutrition) carry the big part of body recovery; NAD+ and other advanced intervention na real but small small thing wey go build on top of dat foundation, e no be replacement for am.
Calcium D-glucarate (200-400mg every day)	Complement to role of NAC above, dey work for different part of liver detoxification: e fit block beta-glucuronidase, enzyme wey fit reverse liver's Phase II conjugation process and make already-packaged toxins and hormone metabolites get reabsorb back into circulation instead of proper way wey e suppose leave. Where NAC support glutathione conjugation pathway direct, calcium D-glucarate help make sure wetin liver don process already actually leave rather than circle round — relevant background support during time when liver dey manage both substance itself and whatever adulterants dey for supply.
Magnesium glycinate (300-400mg elemental, evening)	Magnesium dey sit direct inside the NMDA receptor channel and physically block excess calcium enter unless cell don depolarize well-well — e be body own natural regulator for exactly di glutamate/NMDA overactivation wey I talk before. Dis na extra, no mean say e replace, e role for Section 8's sleep support below.
P5P (pyridoxal-5-phosphate — na active form of vitamin B6, 20-50mg every day)	P5P na cofactor wey e need pass go glutamate decarboxylase (GAD), enzyme wey dey change glutamate to GABA. Without enough P5P, that change no go happen well-well, leave plenty glutamate and less inhibitory GABA counterbalance — direct link to the glutamate-heavy picture say dis section talk about. Use active P5P form instead of regular B6 (pyridoxine) if methylation na worry, according to MTHFR row below.
'Brain Stack' — Uridine + DHA + CDP-Choline	Directly target d2 receptor density upregulate — na structural recovery of the dopamine system self, no be just symptom relief.
5-MTHF (methylated folate)	No be say you fit play with foundational methylation support, especially important if na MTHFR-linked depression/flatness pattern for Section 6 dey apply.
Electrolyte and water, food wey get plenty protein when hunger start come back	E dey directly support crash phase for Section 2; di body no food depletion after binge na real thing, e sabi take like say e be same level with di mind sickness.

⚠ KAIUTION

On ketamine specifically, since e dey come up naturally alongside glutamate: na genuine research interest for ketamine for stimulant use disorder, working through NMDA-related synaptic plasticity rather than by 'reversing' toxicity directly, like how e work for its mechanism for treatment-resistant depression. E be Emerging-tier

evidence, specialist-supervised only, and need real caution pass anything wey make you chase here specifically — the same Reward Deficiency Syndrome vulnerability we discuss throughout Section 6 wey make meth sweet so much fit make second dissociative substance sweet too. Route of administration matter plenty for this: IV ketamine and esketamine (Spravato, intranasal, FDA-approved for treatment-resistant depression — an off-label use if apply to stimulant use disorder specifically) get much higher and more reliable bioavailability pass oral ketamine, including troches — oral routes lose plenty drug to first-pass liver metabolism, make effect no too predictable. For this early, still-repairing window, the more reliable IV or intranasal routes generally consider am the more clinically worthwhile option if person decide follow this path at all; oral ketamine na more common talk for later stage option, closer to one-year mark once the acute dopamine and glutamate repair work for Section 3 don pass much. Dis na talk wey need psychiatrist wey sabi both stimulant use disorder and ketamine's research base, no be say you wan add am for dis stack by yourself.

Tier 2 — Support Wey I Fit Give For Body and Mind

- No be say dem get any medicine wey FDA approve specially for methamphetamine withdrawal — unlike di benzo or SSRI guide, dis level na talk about watchin and support pass chemical way o.
- Psychiatric evaluation when person dey for crash phase e good if depression too heavy, because of real suicide risk time wey dey for Day 1-5 — na no be overreaction, na proper answer to risk wey don show.
- If doctor introduce any medicine (sometimes bupropion dey use off-label for stimulant use disorder), show CYP2D6/CYP2B6 status if you know am, according to Section 6.

Tier 2.5 — 'Ghost Scaffolding': Psychiatrist-Supervised Stimulant Stabilization

Dis one deserve its own honest talk pass just one bullet, because na real way wey dey use for specialist addiction medicine, and e easy too people misunderstand say na 'just get prescription for same kind drug.' E no be dat, when do am correct — but the difference matter plenty die to walk through well-well.

⚠ KAUTION

Dis no be self-directed strategy. E need psychiatrist ma o, or addiction medicine specialist wey dey actively manage decision, monitor for misuse risk, and treat am like time-limited clinical tool — e no be open-ended replacement. To follow stimulant prescription informally, or from general prescriber wey no know full history, na totally different thing and much more risky pass wetin I go talk below.

The clinical logic: di Gray and Slow Return phases for Section 2 dey produce real, measurable executive-function impairment — attention, working memory, task initiation — wey fit look like ADHD pass, whether or no ADHD dey before meth use

start. For some specialist setting (dis one na documented practice for some country's addiction medicine program, sometimes dem call am psychostimulant substitution or agonist-style stabilization), e dey use carefully supervised, long-acting prescription stimulant — extended-release amphetamine salts (Adderall XR) or similar — short-term specifically to bridge dat executive-function gap while di dopamine system for Section 3 do its slower underlying repair work, for di like use buprenorphine to steady up opioid-dependent system instead of just leave am to recover all by itself.

Wetin dis be?	Wetin dis no be
Time-limited, specialist-supervised clinical tool wey make e focus on the specific executive-function gap for Section 2's Gray/Slow Return phases.	Green light make you dose yourself when you fall down with any stimulant, whether doctor prescribe am or no.
Use am with, no be pass, di Tier 1 and Tier 3 work for dis section	Substitute passin' di exercise, sleep, and neuroplasticity work wey really drive di D2 receptor recovery for Section 3
Ebi wey no get pass evidence abi e dey debate well-well inside addiction medicine — evidence tier: Emerging, no be Established	One standard wey dem sabi well-well — most addiction specialist still dey use am with caution, or no use am at all.

Kedu be say you dey hear?

If na fog and flatness for early recovery dey make you wonder if you really get ADHD, or if e always dey there — dat one be legitimate question wey you need raise with psychiatrist, no be thing wey you fit just dismiss as 'just withdrawal talking.' Sometimes e be withdrawal. Sometimes real, previously undiagnosed ADHD na part of wetin make stimulants sweet so much for first place, according to Section 6's phenotyping questions. Specialist fit actually help you tell the difference, which na exactly conversation say 'ghost scaffolding' mean to open, no be replace.

Tier 3 — Support for Nervous System and Neuroplasticity (The Repair Signal)

- Exercise — na e be pass important thing for whole protocol dis one. BDNF, signal wey go repair damaged dopamine axons wey dem talk about for Section 2 and Section 3, na say e come mostly from physical activity. Start as soon as body fit do am, even small time, and build up to 30-45 minutes of moderate-to-high intensity almost every day.
- Sleep — 7-9 hours, schedule wey no dey change; BDNF synthesis na so e go high and memory consolidation too, e mean say sleep wey dey change go slow down the repair process wey talk for Section 3.
- Sunlight morning — 20+ minute dey outside, best time na inside first two hour after you wake up, abi make your body clock align well-well and give you good mood.

- Novel, challenging learning activity — 30+ minute every day of something wey e hard to learn (instrument, language, complex skill) go drive neuroplasticity pass passive things.
- Structured daily routine na wetin go help, plus change environment — remove or alter trigger wey dey follow location, person, and thing wey you use.

Section 9 — On Stigma

Methamphetamine carry kind heavy weight of social stigma pass most other substance wey dey for this library — the 'tweaker' stereotype, assumption say person be criminal or e don fail morally, and level of disgust way dem talk about drug and user publicly say alcohol or even opioid dependence no carry to same degree. E good make we name am straight rather than leave am silent, because e get real, documented practical effect: people with methamphetamine use disorder report — and research on healthcare-provider bias confirm — dey take care pass, pain management no too thorough, and treatment dem give dem say na joke. from medical provider pass people wey get other substance use disorder, even when dem show same symptom.

Kedu be say you dey hear?

If you don feel say you need to prepare yourself before talk to doctor, dentist, or ER wetin dey happen—that feeling no be paranoia. E be proper reaction to real pattern. E no mean say every provider go treat you like dat, and e no mean say you suppose hide information wey dey keep you safe (for Section 0 and Section 5's psychosis-risk overlap, correct history important for your own care)—but the caution wey you carry into that talk na no overreaction to nothing.

- If tone person wey dey give you start shift well-well when dem hear say you use meth, na thing wey you fit talk direct to dem ('I wan make sure say this history no go change quality of care I dey get here') or, if dat one no work, e good to raise am to different provider — you sabi do dat.
- Adiction medicine specialist and clinic wey dey focus on harm reduction, dem usually no get dis kind bias inside like general practice or emergency setting — e good to look for am specially if you need continuous care and you fit reach am.
- Stigma na say drug no good, e no concern with you personally. Ebi say dopamine system rebuild wey dem talk for Section 3, e no care wetin anybody think about your character while e dey do that work.

Section 10 — Recognize Danger Again

⚠ KAIUTION

Call 08062106493 or 112 sharp sharp for: thought say you wan die or plan specific one, chest pain or heart beat dey wrong, headache wey sudden come with signs of stroke (face don drop, arm weak, talk no clear), psychosis (wahala mind wey no true, see thing wey no dey), or agitation pass level wey make person lose control. For the crash symptoms alone — tire, sleep too much, depression, craving, brain fog — na wetin expected according to Section 2, but support contact and, if e serious, medical or psychiatric check-up same day still good.

Section 11 — Full Protocol Cross-Reference Map

Reference	Wetin e give here
Recovery Kernel Meth/Stimulant Guide, Section 3 (The Detox Protocol)	Whole timeline wey dis guide's Section 2 build from, plus whole supplement dosing and timing detail behind Section 8's Tier 1.
Recovery Kernel Compendium, Section 16.4 (Receptor Recovery — The Evidence)	Di citation na Volkow et al. and Heinz et al. wey dey support di 12-17 month timeline for Section 3, plus di comparative data for alcohol and cocaine receptor recovery.
Recovery Kernel Compendium, Protocol 01 (Shelter)	Di crisis-stabilization framework wey dem refer to through out Section 2's crash and pit phases.
Recovery Kernel Compendium, Protocol 05 (Environmental Redesign)	Di trigger-remove na e, and di environment-change work wey dey talk for Section 8, Tier 3 — identify and change people, place, and thing wey relate to using.
Recovery Kernel Compendium, Protocol 06 (Daily Structure)	Di consistent-routine framework e fit start well-well for Week 2 to Week 4 for Section 2, when di sharp crash don pass.
Recovery Kernel Compendium, Protocol 20 (Identity Reconstruction) and C-28 ('The Recast')	Shine start for Month 1-3 for Section 2 — no before dat. Build identity and set of real, strong things wey fit person wey get Warrior COMT profile for Section 6, instead of one wey revolve round drug or say e no dey.
Recovery Kernel Compendium, Section 16 (Neuroplasticity Architecture)	Di whole 90-day rebuild framework wey dem talk for Section 3 — di exercise, sleep, learning, and sunlight protocol with all details, including di week structure behind Section 8's Tier 3.
Recovery Kernel Chemsex Guide	If methamphetamine use happen for chemsex context specially — na the combined GHB/meth/mephedrone recovery timeline and unified supplement stack dey address di multi-substance picture dis guide cover only for meth alone.
Recovery Kernel Complex Trauma and the Substance Connection	For dis guide's own Section 3 (Substance-Trauma Co-Axis): chronic hypervigilance wey e turn to force make person achieve, na one documented path wey come from trauma go lead to stimulant use specifically — e good make you read am alongside this guide if dat pattern dey follow you.

Section 12 — Closing

IBELE

Dopamine system wey methamphetamine hijack am no mean say e don break forever. Data tin specific pass and true: recovery wey you fit measure within few week, structural recovery wey strong well-well within year to year and half, and 90 days window where deliberate exercise, sleep, and novel challenge do more rebuilding work pass any other time for process. The wahala of the crash no ever be sign say something don go wrong. E be, and e still be, signal say repair don start already.

END OF GUIDE — DISCONTINUANCE PROTOCOL: METHAMPHETAMINE

Harm Reduction Resource — E no be Medical Advice — E no be Replacement for Emergency Care

Crisis Resources: 988 (Suicide & Crisis Lifeline, call or text) | Crisis Text: HOME to 741741 | SAMHSA: 1-800-662-4357